

9-1-2015

Do the Perceived Cognitive Enhancing Benefits of Caffeine Outweigh the Risks to Mental Health?

Stephanie Brezinski
Winona State University

Follow this and additional works at: <https://openriver.winona.edu/studentgrants2016>

Recommended Citation

Brezinski, Stephanie, "Do the Perceived Cognitive Enhancing Benefits of Caffeine Outweigh the Risks to Mental Health?" (2015). *Student Research and Creative Projects 2015-2016*. 2.
<https://openriver.winona.edu/studentgrants2016/2>

This Grant is brought to you for free and open access by the Grants & Sponsored Projects at OpenRiver. It has been accepted for inclusion in Student Research and Creative Projects 2015-2016 by an authorized administrator of OpenRiver. For more information, please contact klarson@winona.edu.

Do the Perceived Cognitive Enhancing Benefits of Caffeine Outweigh the Risks to Mental Health?

Research Summary

Stephanie J Brezinski

Department of Psychology

Winona State University

SBrezinski12@winona.edu

Purpose

Caffeine is a psychostimulant commonly used to alleviate drowsiness and increase alertness. Almost everyone consumes caffeine on a daily basis through drinks (e.g., coffee, tea and energy drinks), food (mainly chocolate), or medications such as diet pills and migraine treatments (Nehlig, 2010). Because caffeine is widely viewed as safe, it is commonly used at relatively high levels among college students to enhance academic performance and cope with the stress of their workload (Kalash and Tannous, 2014). It has long been known that high caffeine consumption produces the condition of caffeinism, which most often presents as an anxiety disorder (MacKay and Rollins, 1989; Strous, 2006; Hedges, Woon and Hoopes, 2009; Roy-Byrne, 2015). Even moderate to low doses can exacerbate existing psychological disorders (e.g., Benke, Blumenthal, Mode, Hamm and Pané-Farré, 2015). Therefore, given these risks to mental health and that 68.5% of WSU students report using moderate to heavy doses of caffeine to enhance academic performance, it has become increasingly important to determine whether the perceived cognitive enhancing benefits of caffeine use outweigh the risks to mental health (Fitzgerald, Glavanovich, Howes, and Krzyanowski, 2010; Garlick, and Luebke, 2013).

Nehlig (2010) reviewed caffeine's effects on learning and found that 64% of those studies reported that caffeine had no effect on performance, 24% of the studies showed caffeine-induced improved free recall and 12% of studies observed caffeine impaired recall. There are several possible explanations for a lack of consensus on caffeine's behavioral effects.

First, it has been impossible to obtain a pure control group (i.e., never used or not currently using caffeine) for human studies of caffeine's psychotropic effects due to its widespread use. In my review of the literature, I was unable to find a single study with a true control group. Researchers commonly use overnight abstinence of caffeine prior to testing to

create the control group, however, the problem is that overnight abstinence follows approximately the same time frame (10-14 h) in which withdrawal symptoms appear. So any reported benefit of caffeine in studies using this design could mainly be due to the reversal of aversive withdrawal symptoms such as: fatigue, difficulty concentrating, decreased energy and headache (James and Rogers, 2005). Second, caffeine has a relatively fast half-life (2.5-4.5 h) but it would still be expected to be in the system for up to 48 hours suggesting that some heavy users may still be defined as caffeine treated even after overnight abstinence. Finally, none of the studies reviewed tested for caffeine levels in the participants but rather used self-report which is likely to produce inaccuracy. These three factors alone question the validity of the control group most commonly used in human caffeine research. This observation led me to design a pilot study in preparation for this grant application in which I replicated the traditional caffeine study with two changes. First, I obtained saliva samples before and after caffeine consumption in both controls and caffeine treated participants and determined salivary caffeine levels. Second, participants were tested twice so that they served in both the caffeine and control groups to control for differences in rates of caffeine metabolism.

I hypothesized that 46 mg of caffeine (found in a single can of diet Coke) would improve college students' learning and memory during free recall testing after abstaining from caffeine for 12 h. Over the course of 48 hours, using a within-subjects, double blind design, I gave nine Winona State students either caffeinated diet Coke (46 mg) or caffeine free Coke zero 30 minutes prior to free recall testing. On both test days, saliva samples were taken prior to beverage consumption to determine if participants abstained from caffeine 12 h before testing as instructed. Participants consumed their beverage, waited 30 minutes for the caffeine to take effect, and then completed five trials of a free recall test. The test consisted of 25 words

individually displayed on the computer screen for 1 s. A 20 s delay was imposed between the presentation of the last word and the actual recall test. Participants were then given 60 seconds to type as many of the words that they could remember. A second saliva sample was collected after the participants completed the fifth trial. An ELISA was completed on the saliva samples to determine the participant's actual salivary caffeine levels. To test whether or not learning occurred, I calculated a difference score by subtracting the number of words correctly recalled during trial one from trial five.

I found a curvilinear correlation between salivary caffeine and free recall. Specifically, caffeine concentrations less than approximately 1500 ppb showed a positive correlation, suggesting that 46 mg of caffeine may be enough to show significant improvement in performance during free recall testing. Additionally, caffeine concentrations between 1500 and 2000 ppb impaired recall. These pilot data suggest a dose-dependent relationship between caffeine and memory in college students, however, only one participant arrived completely caffeine free even though all students reported abstaining from caffeine. This means, like previous studies, my control group was also confounded. Therefore, the method of using overnight abstinence as a control group is inappropriate and a different research design is required to determine caffeine's psychotropic actions. Therefore, the purpose of the experiment outlined in this proposal was to use a true caffeine free control group in order to evaluate the effects of caffeine use on learning and memory as well as determine whether cognitive enhancing benefits can be obtained without the potential for adverse psychotropic effects such as anxiety.

Methods

Participants

Eighteen participants were recruited from upper level psychology courses and randomly assigned to either the experimental group (receiving 46 mg of caffeine from a 12 oz. diet Coke) or the control group (12 oz. of caffeine free Coke Zero). Persons taking anxiolytics, antidepressants, neuroleptics, mood stabilizers or migraine medications were excluded to control for potential drug interactions as each of these drugs can change the net dose of caffeine by altering its metabolism.

Materials and Procedures

A laptop equipped with the testing software developed in-house was used. Over the course of eight days, saliva samples, self-evaluations of withdrawal symptoms and physiological and psychological measures of anxiety were collected and recorded using the protocol outlined in Table 1. Days zero through four used self-evaluations to record withdrawal symptoms participants experienced during caffeine abstinence. On day five, test day, each participant received 12 oz. of the assigned beverage, waited 30 minutes for the beverage to take effect, and then completed a test of learning and memory. Oximeters and self-evaluations were used to record both physiological and psychological symptoms of anxiety post beverage consumption on days five through eight.

On the test day, a practice trial was completed to familiarize participants with the testing procedure. Participants then completed five trials each consisting of the presentation of the same 25 words individually shown for 1 s followed by a 20 s delay during which participants were required to memorize the words. Participants were then allowed 60 s to type as many words as they could remember. To determine at what rate each participant metabolizes caffeine, the researcher collected additional saliva samples at the following times post test: 2 h, 4 h, 24 h, 48 h and 72 h. Once all of the data was collected, the researcher completed a caffeine ELISA kit to

test salivary caffeine levels at day zero, day one, day five (pre-treatment, +45 minutes, +2 hours, +4 hours), +24 hours and +48 hours for all participants.

Table 1. Summary of Research Protocol

Day	Saliva Collection/ Symptom Assessment	Day	Saliva Collection/ Symptom Assessment
0	Initial collection, commit to abstinence	5b.	Post-memory test sample (45 mins)
1	24 h abstained	5c.	Post-test sample (2 h)
2	48 h abstained	5d.	Post-test sample (4 h)
3	72 h abstained	6	Post-test sample (24 h)
4	96 h abstained	7	Post-test sample (48 h)
5	Memory Test Day 120 h abstained/Pre-beverage sample	8	Post-test sample (72 h)

Results

Figure 1 A illustrates the mean salivary caffeine levels at day zero and at day five. As predicted, salivary caffeine levels changed significantly from day zero to day five, $F(1, 14) = 10.428$, $p < .01$. However, gender did not significantly effect salivary caffeine levels $F(1, 14) = .010$, $p = .922$. Figure 1 B illustrates the mean salivary caffeine levels at day zero and day one. When analyzing if drug condition and gender had an effect on pulse at 120 hours abstained (day five) and 45 minutes post-drug treatment, the results were as follows. Drug condition did not significantly effect pulse at 120 hours abstained ($F(1, 14) = 3.918$, $p = .068$). However, caffeine significantly reduced pulse at 45 minutes post-treatment, ($F(1, 14) = 9.518$, $p = .008$). Additionally, gender did not significantly effect pulse at both 120 hours abstained, ($F(1, 14) = 3.712$, $p = .075$) and 45 minutes post-drug treatment ($F(1,14) = 4.196$, $p = .060$). Lastly, there is no significant interaction effect of drug condition and gender for pulse at 120 hours abstained, ($F(1,14) = .034$, $p = .989$) and at 45 minutes post-drug treatment, ($F(1,14) = .163$, $p = .970$).

When analyzing intoxication scores at baseline (day 0), no significant effect was found for either drug condition, ($F(1, 10) = .005$, $p = .943$) or gender ($F(1, 10) = 2.119$, $p = .176$). Also, there is no significant interaction between drug condition and gender, ($F(1, 10) = .339$, $p = .573$). These results suggest that intoxication scores at baseline were not influenced by the drug condition assigned or gender. Acquisition did change significantly from trial one to trial five, ($F(1, 14) = 136.175$, $p = .000$). However, caffeine treatment did not significantly effect acquisition ($F(1, 14) = 1.623$, $p = .223$) nor was there a significant gender effect ($F(1, 14) = 3.897$, $p = .060$). Memory retention did not change significantly over time (2, 4, 24 and 48 hours) after the memory test ($F(1, 14) = .323$, $p = .579$). Additionally, caffeine treatment did not significantly effect retention, ($F(1, 14) = 3.896$, $p = .068$) nor was there a significant gender effect ($F(1, 14) = 2.768$, $p = .118$). Withdrawal ratings did not change significantly over time (2, 4, 24 or 48 hours after treatment); $F(1, 14) = 1.524$, $p = .237$). Additionally, caffeine treatment did not significantly effect withdrawal ratings ($F(1, 14) = 1.581$, $p = .229$) nor was there a significant gender effect ($F(1, 14) = .806$, $p = .384$).

Discussion

The purpose of this experiment was to use a true caffeine free control group in order to evaluate the effects of caffeine use on learning and memory as well as determine whether cognitive enhancing benefits can be obtained without the potential for adverse psychotropic effects such as anxiety. The present study showed that participants still had significant amounts of caffeine in their systems after 24 hours of abstinence (more than double the abstinence period used in prior studies). The data also showed that all participants in the present study had salivary caffeine levels reduced to zero after five days. Prior to the completion of the current study, most caffeine research used overnight abstinence as a research design, but the data found in this study

(see Figure 1) clearly demonstrates that overnight abstinence is an inappropriate method and a five-day washout, as used in our study, is required to obtain a true control group for studies testing the effects of caffeine. Unfortunately, these data seriously call into question the validity of most if not all previous caffeine research with human participants.

Although we successfully reached our initial object of testing at least 20 subjects, we have elected to continue running additional participants to improve several aspects of our study. First, there were not enough male volunteers ($n=4$) who participated in this study to fully evaluate possible gender effects. Therefore, one of our goals moving forward with this study will be to increase the number of male participants. Additionally, a number of our female participants reported using oral contraceptives, which research shows can affect the metabolism rate of caffeine. At present we do not have enough female participants using the various forms of contraception to fully control for possible confounding effects of these drugs on caffeine metabolism. Thus, our second goal this fall will be to control for the effects of different methods of birth control (i.e. oral, injection, subdural) on caffeine metabolism by specifically recruiting females from these subgroups. Our plan will be to continue running participants throughout the summer of 2016 through fall semester 2016. We recently submitted a request to the WSU IRB to extend testing and approval was granted on 5-11-16.

If accepted, we plan to disseminate the preliminary data collected through May 10, 2016 at the annual Midbrains Neuroscience Research Conference in October 2016. When all of the subgroups are adequately represented we plan to submit the paper for consideration for publication. Copies will be made available. We wish to thank WSU Research Grant fund.

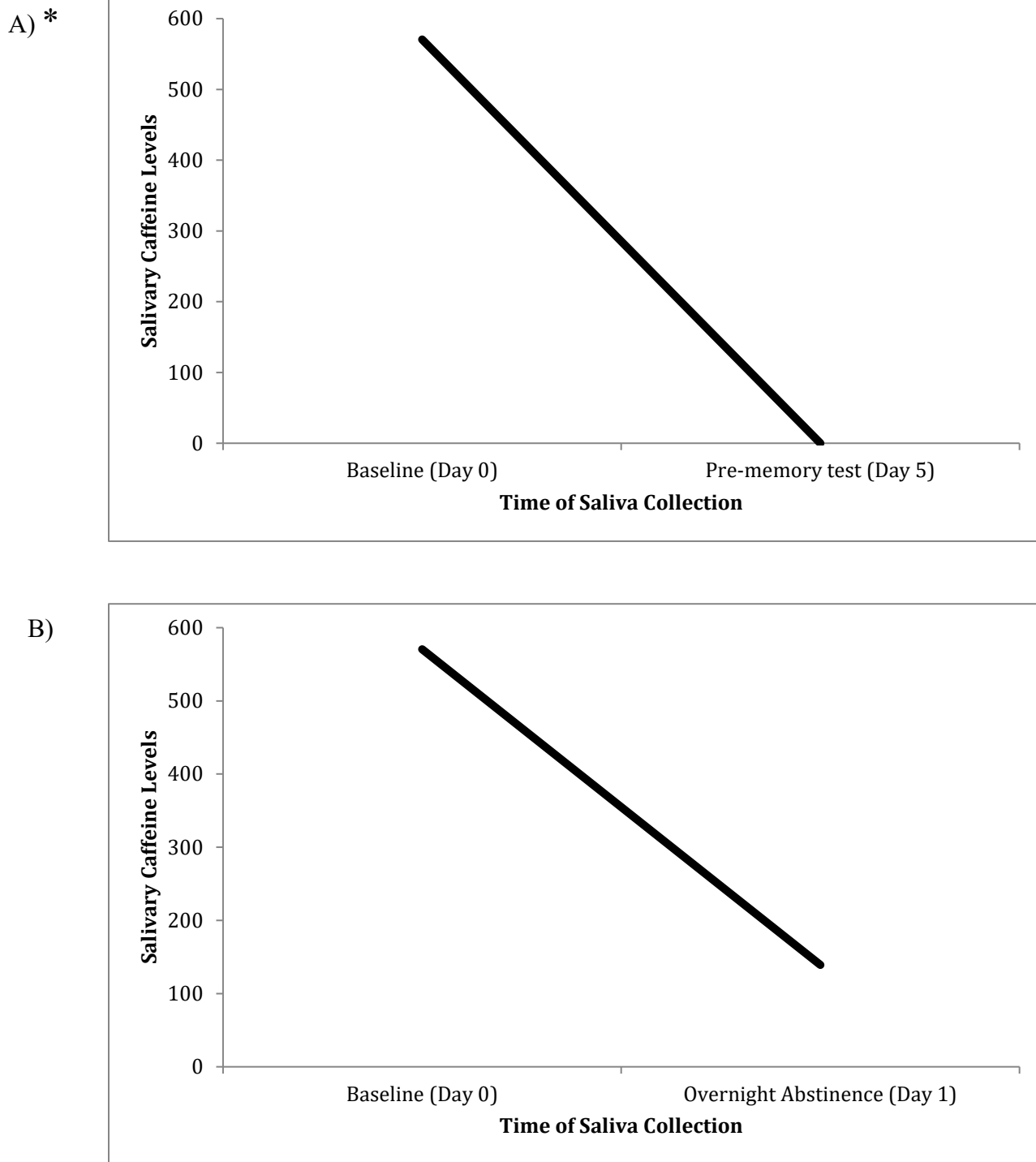


Figure 1. Summary of the mean salivary caffeine levels at A) baseline and pre-memory test, and B) baseline and overnight abstinence. * Indicates significance at the .05 level.

References

- Benke, C., Blumenthal, T. D., Mode, C., Hamm, A. O., and Pané-Farré, C. A. (2015). Effects of anxiety sensitivity and expectations on the modulation of the startle eyeblink response during a caffeine challenge. *Psychopharmacology*, 232, 3403-3416.
- Fitzgerald, M., Glavanovich, N., Howes, N. and Krzyanowski, B. (2010). Survey of college drug use: is Deja-vu (JWH018) a new trend? Presented at the 20th Annual Tri-State Undergraduate Psychology Research Conference. Loras College, Dubuque, IA.
- Garlick, L. and Luebke, C. (2013). Use of JWH018 and marijuana by college students. Presented at the 7th Annual Mid Brains Undergraduate Neuroscience Conference of the Midwest. Carleton College, Northfield, MN.
- Hedges, D.W., Hoopes S.P. and Woon F.L. (2009). Caffeine-induced psychosis. *CNS Spectr.*, 14, 127-9.
- Iancu, I. and Strous R.D. (2006). Caffeine intoxication: History, clinical feature, diagnosis and treatment. *Harefuah*, 145, 147-51, 163-4.
- James, J. E. and Rogers, P. J. (2005). Effects of caffeine on performance and mood: Withdrawal reversal is the most plausible explanation. *Psychopharmacology*, 182, 1-8.
- Kalash, Y., and Tannous, M. (2014). Prevalence of caffeinated-beverage consumption by university students in north Lebanon. *Public Health Research*, 4, 173-178.
- Mackay, D.C. and Rollins J.W. (1989). Caffeine and caffeinism. *J R Nav Med Serv*, 75, 65-7.
- Nehlig, A. (2010). Is caffeine a cognitive enhancer? *Journal of Alzheimer's Disease*, 20, S85-S94.
- Roy-Byrne, P. (2015). Treatment-refractory anxiety definition, risk factors and treatment challenges. *Dialogues in Clinical Neuroscience*, 17, 191-206.

Final Budget

Item(s)	Amount
Data Analysis:	
ELISA kits (3 x \$500 each)	11/05/15 - \$570.00
Testing Supplies:	
Diet Coke and Coke Zero	\$30.00
Plastic Cups	
Total Expenditures:	\$600.00

WINONA STATE UNIVERSITY
UNDERGRADUATE STUDENT RESEARCH & CREATIVE PROJECTS FINAL REPORT

*Electronically submit complete final report ten (10) days following completion of project to Grants & Sponsored Projects (grants@winona.edu).
Hover over fill-able fields for additional guideline and completion information.*

Student Name:

Student Email:

Student Major:

Faculty Sponsor:

Faculty Sponsor Email:

Title of Project:

Project Abstract:

The student-authored final report **MUST** include each of the following (check boxes to verify inclusion of each component):

This report form, fully completed (page 1 of this form)

A copy of the project end product, appropriate to the standards of the discipline

Date:

Applicant Signature:

Date:

Faculty Sponsor Signature:

Submit complete reports electronically to Grants & Sponsored Projects (grants@winona.edu).

A deans' sub-committee makes decisions on Undergraduate Student Research & Creative Projects proposals.

Note that a copy of the project end product will be forwarded to Krueger Library for archival purposes.